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REMARKS

Claims 19 and 21 - 35 are still pending.

In the Office Action, the rejections of claims 19 and 21 – 35 under 35 USC §112, first paragraph, as well as under §102(e) and §103, were maintained. These rejections are again respectfully traversed, as further explained and argued below.

The Office Action states that the rejections are being maintained because Applicants "did not provide a structure of Guanidine acetate to show that it is not structurally equivalent to Guanidinoacetate." (Office Action, page 2, first paragraph.)

In Appplicants' previous response, it was stated:

"The comments in the Office Action appear to indicate some confusion about the molecules 'guanidinoacetate' and 'guanidine salt'. In the previous response, Applicants stated that "...'guanidine acetate', ...may be recognized as a salt of guanidine...". In fact, **Applicants are unaware if any such salt of guanidine exists**. However, the statement was only intended to point out an apparent discrepancy in nomenclature, which is that *guanidinoacetate* is not equivalent (in chemical name) to *guanidine acetate*. To further illustrate the differences in the compound of Kaddurah-Daouk (guanidinoacetate) and a guanidine salt according to the present invention, attached to this paper (as Exhibit A) is a diagram showing the chemical structures of guanidine (showing also its ionic (base) form), and guanidinoacetate. Despite sharing the root "guanidin-", they are clearly different molecular entities. In fact, guanidinoacetate (GAA) is a salt or anionic form of *guanidinoacetic acid* (identical to the structure of guanidinoacetate shown, but with an H atom at the COO group). Structurally, the GAA contains an additional C atom and the acetyl moiety as compared to guanidine. Moreover, chemically they are quite different, because guanidine is a strong base and GAA is an acid form. Exhibit A also contains a list of a number of commonly available guanidine salts and their structures. **No mention of 'guanidine acetate' could be found in the literature.**" [May 10, 2006 Response, emphasis added.]

Of course Applicants could not have provided a structure of "guanidine acetate", because **no such chemical compound exists**. As stated above,

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guanidine is a strong base and as such cannot form a salt with an acetate moiety. Applicants urge that this matter be put to rest on the basis of these facts, and that the rejections under §112, §102 and §103, be withdrawn.

Claims 30 and 32 were rejected as being indefinite under §112, second paragraph. These claims have been amended to clarify any possible misunderstanding as to what is being administered. Withdrawal of this rejection is deemed appropriate.

The rejections of claims 19, 23-29 and 31 under §102(b) and of claim 32 under §103(a) over Azumendi (GB 2315672) are respectfully traversed.

The Office Action states that Azumendi teaches treating prion diseases in mammals by administering an effective amount of NaI or KI to the mammals. (The aspect of hyperthermia being induced by these agents is not understood. These agents do not cause hyperthermia, and no where in Applicants' specification or claims is this disclosed. Hyperthermia is an optional, additional component of treatment in accordance with the present invention.)

It is respectfully submitted that the UK Patent Application publication of Azumendi cannot be interpreted as disclosing a method of treating prion diseases by administering NaI or KI.

Azumendi's disclosure relates to the treatment of protozoa cysts, such as those of Sarcocystis protozoa, which cause the condition "Sarcocystosis" in mammals. According to Azumendi, page 4, 27 – 29, Sarcocystis infection results in muscle spasms, diarrhea and chronic fatigue, and speculates that, since those

symptoms are also present in demylinating diseases such as multiple sclerosis, then such demylinating diseases must also be caused by Sarcocystis infection (page 4, lines 30 – 32), and possibly by an isolated toxin of the Sarcocystis protozoa (page 4, lines 32 – 35). Azumendi then theorizes that such demylinating diseases could also be treated by treating the *supposed* protozoal infection.

Further, at page 5, lines 9 – 20, Azumendi proposes that, because of a "close *similarity of characteristics* between the...prion and the toxin", they must be one and the same. Thus, speculates Azumendi, the treatment disclosed will also be effective in treating prion diseases such as BSE and CJD. However, Azumendi does not disclose what these similarities are between the toxin and prion protein. He does not mention anything about structural or similarities, and it can only be reasonably inferred that Azumendi is referring to supposedly similar effects caused by the two agents.

In other words, Azumendi is concluding that prion diseases are caused by Sarcocystis infection. In fact, Azumendi's entire disclosure and claims require the presumption that what is being treated is the protozoal infection itself. (It should be kept in mind that Azumendi is only a publication of an unexamined application.) The hypothesis that protozoal infection causes diseases such as CJD or BSE has never been proven, and it is not a view held by scientists in the area of prion diseases. At the time of the present invention, it was generally accepted that the prion protein is the causative agent of prion diseases such as

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CJD. Protozoal infection has never been shown to be the cause of prion diseases.

Accordingly, the rejections under §102(b) and §103(a) should be reconsidered and withdrawn.

Finally, claims 23 and 27 have been amended to address the objections to these claims.

Applicants respectfully submit that claims 19 and 21 – 35 are in condition for allowance. Given the protracted prosecution of this application, Applicants are submitting herewith a Notice of Appeal, and request an appeal conference in order to expedite the disposition of this application.

A Petition for one-month extension of time is also submitted with this paper.

Respectfully submitted,

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Enclosures: Petition for Extension of Time
Notice of Appeal